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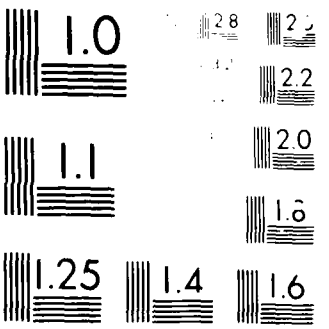
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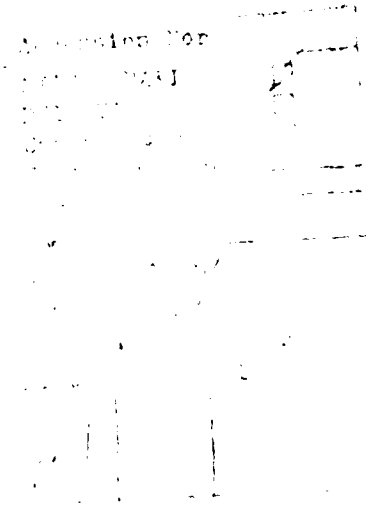
Slowly-Developing Modifications in Cutaneous Circulatory Control

C. Bruce Wenger

U. S. Army Research Institute of Environmental Medicine, Natick, MA 01760

and Department of Environmental Physiology, Harvard School of Public Health,

Boston, MA 02115



The factors discussed below affect skin blood flow (SkBF) through nonthermal mechanisms. In considering them, however, it is necessary to account for thermoregulatory effects which may operate at the same time, because of the dominant role of the latter in the control of SkBF. Therefore, these non-thermal factors will be approached in terms of how they modify the relation of forearm blood flow (ABF), probably the most widely used index of SkBF, to the thermoregulatory signal, as represented by esophageal temperature (T_{es}) when skin temperature is not changing. Slowly developing modifications may have time courses ranging from minutes (e.g., transcapillary fluid shifts, peripheral pooling of blood) through hours (e.g., circadian rhythms, dehydration) to weeks (e.g., exercise training, heat acclimation, menstrual cycle). Most of these factors change the threshold for vasodilation, but some also change the slope of the ABF:T_{es} relation.

Several of the factors that change the threshold for vasodilation likewise change thresholds for other thermoregulatory responses, such as sweating. If the operation of the thermoregulatory system as a whole is shifted to a different temperature level, such a shift represents a change in thermoregulatory "set-point" (7). Fever was probably the first condition to be recognized as resulting from a change in thermoregulatory set-point, and as expected, the threshold for forearm vasodilation is elevated in fever (11). Other examples of set-point change are the circadian (19) and menstrual cycle (9) rhythms in body temperature. Recent studies of both of these rhythms illustrate quite nicely the criterion that a set-point change affects other thermoregulatory responses the same as SkBF (9,19).

Other factors that appear to change the thermoregulatory set-point are endurance training and heat acclimation. Long distance runners have lower thresholds for both sweating and shivering than do controls (1), and both endurance training and heat acclimation lower thresholds for sweating and vasodilation (16).

However, threshold shifts for vasodilation averaged twice as great as those for sweating (16), in contrast to the closely matched threshold shifts for vasodilation and sweating during the circadian and menstrual cycles (9,19). Training and acclimation seem thus to involve not only thermoregulatory changes, but also specifically cardiovascular effects, probably related to the plasma volume (8) and myocardial (e.g. 10) changes reported to accompany training and acclimation.

Factors that affect the control of SkBF specifically, without changing the thermoregulatory set-point, may operate through baroreflexes, chemoreflexes, and mechanisms related to electrolyte concentrations and osmolality. Although thermoregulatory reflexes work through both vasoconstrictor and vasodilator nerves (6), these non-thermal effects are only known to work via vasoconstrictor fibers, since they appear not to increase ABF above the levels observed at thermoneutrality. However, in warm vasodilated subjects they may also inhibit outflow of vasodilator signals.

Both arterial and cardiopulmonary baroreflexes seem to affect SkBF (2), and there is no ready way to separate their effects in the following studies. Fig. 1 shows data obtained on subjects exercising in the supine (SX) and upright (UX) postures (15). At any point during UX, cardiac stroke volume (SV) and ABF were lower, and T_{es} higher than during SX. ABF was thus lower at any T_{es} during UX, and the threshold for vasodilation was higher. As SkBF increases, so do peripheral venous pressure and, presumably, pooling of blood in the compliant skin veins (17). Gravity favors such peripheral pooling during UX, so that SV is lower than in SX, and drops further as ABF increases. (During SX, by contrast, SV falls rather little, even though ABF rises much higher than in UX.) The lower SkBF during UX than during SX tends to limit peripheral pooling and keep SV from falling even more.

Another factor that probably contributes to lowering SV during exercise is the decrease in blood volume owing to the shift of water from plasma to the interstitial space (e.g. 12). Similar reductions in blood volume, produced by blood withdrawal (4) or diuretic treatment (13) before an experiment, reduce stroke volume and raise the threshold for vasodilation during exercise. Besides threshold elevations, another modification that may occur in the ABF:T_{es} relation, especially during exercise with reduced BV in the heat, is a "break point", above which the slope of the relation is markedly reduced (3,12). Such break points are not intrinsic to the thermoregulatory control of ABF, and can be eliminated by maneuvers, like supine posture, that favor cardiac filling (12).

Both blood withdrawal and diuretic treatment reduced BV without changing plasma osmolality. In thermal dehydration, however, plasma osmolality increases as BV falls. To investigate the effect of osmolality on the control of ABF, subjects were dehydrated by intermittent light exercise in the heat until they lost 3% of their body weight (5). They then rested in a cool room (and, in some experiments, were infused with a small volume of 3% saline solution) for 1 h, and afterward underwent an exercise test. Dehydration alone raised plasma osmolality by 10 mosm/l above control and reduced BV by 200 ml, and dehydration with infusion raised osmolality by 11 mosm/l without affecting BV. During the subsequent exercise tests, the threshold for vasodilation was elevated in both experimental conditions, as compared with control, and dehydration without infusion also reduced the slope of the ABF:T_{es} relation. Since dehydration changed BV so little, the accompanying changes in the control of ABF do not seem necessary to preserve cardiac filling, and their function in homeostasis is not clear. The control of sweating was affected similarly to that of ABF, but it is unlikely that plasma osmolality affects the thermoregulatory setpoint, since levels of T_{es} at the start of the experiment differed little among conditions, and Nielsen reported

that osmolality affects neither core temperature at rest nor thermoregulation during exercise in a cool environment (14). Such similar effects on the control of ABF and sweating without a change in thermoregulatory setpoint may support the hypothesis of a mechanistic link between sweating and vasodilation (6).

Kolka, Stephenson, Gonzales, and Rock (personal communication) got ABF:T_{es} relations on subjects exercising at normal atmospheric pressure and 552 and 428 torr. Exercise intensities were adjusted so as to be the same fraction of the subject's maximum O_2 consumption for that pressure, so that heart rate and increase in T_{es} were independent of pressure. At lower pressures, the slope of the ABF:T_{es} relation was reduced and the threshold for vasodilation was slightly but significantly lower. Rowell and his colleagues found an equivocal effect of hypoxia on ABF in two of four subjects who began breathing a hypoxic gas mixture during exercise (18). The slope and threshold changes found by Kolka et al. would tend to oppose each other and have little net effect on ABF at T_{es} above threshold. Although it is tempting to interpret such effects on ABF as part of a chemoreceptor-mediated pressor response, other factors may have played a role, since plasma norepinephrine was higher at lower pressures, and the subjects were probably hyperventilating and thus hypocapnic at the lower pressures.

In conclusion, several factors modify the control of SkBF along with that of the other thermoregulatory responses by changing the thermoregulatory setpoint. Other factors, associated with exercise and cardiovascular strain, affect control of SkBF selectively and serve to maintain cardiac filling and muscle perfusion. Finally, the changes in the control of SkBF that accompany hyperosmolality of the blood seem neither to be part of a change in thermoregulatory setpoint nor to serve any apparent cardiovascular function.

The views, opinions, and/or findings contained in this report are those of the author and should not be construed as an official Department of the Army position, policy, or decision, unless so designated by other official documentation. Approved for public release; distribution unlimited.

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FIGURE CAPTION

Fig. 1. Esophageal and mean skin temperatures, cardiac stroke volume, and forearm blood flow during two 20 min bouts of cycle exercise at 41% of maximal O_2 consumption in 40°C ambient temperature. Blood flow is plotted at 1-min intervals for clarity. From Roberts and Wenger (15), with permission of the American Physiological Society.

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